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Cerebral Air Emboli With Atrial-Esophageal Fistula Following Atrial Fibrillation Ablation: A Case Report and Review

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Abstract

Background: Atrial-esophageal fistula (AEF) is a rare and early complication of radiofrequency ablation for medically refractory atrial fibrillation, but has devastating consequences when the diagnosis is delayed or difficult to make. **Methods:** Single case in a neurosciences critical care center. **Results:** A 69-year-old man with significant cardiac and neurologic medical history who underwent atrial fibrillation ablation 50 days prior to admission to the neurocritical care unit presented with acute left-sided weakness and gram-positive bacterial sepsis. This is an exceptional case discussing the need for early detection of AEF presenting with sepsis, neurologic deficit along with complicated decision-making in the neurocritical care setting. His hospital course was complicated by acute stroke, left ventricular (LV) aneurysm with thrombus, gastrointestinal (GI) bleed discovered to be from left atrial esophageal fistula, and subsequent cerebral air emboli leading to death. **Conclusions:** This is the most delayed presentation of AEF following atrial fibrillation ablation reported in the literature to date. We emphasize the need for awareness of this complication even after such an unexpected time-frame postprocedure as well as the unintended complications of cerebral air emboli following upper endoscopy.

Keywords

atrial fibrillation, atrial esophageal fistula, AEF, left ventricular aneurysm, cerebral air emboli

Introduction

Atrial-esophageal fistula (AEF) after radiofrequency ablation is a rare but often fatal complication. Neurologists need to be aware of this complication in patients with new neurological deficits and recent history of ablation for atrial fibrillation. Indirect injury to the esophagus secondary to elevated esophageal temperature has been reported to occur in nearly 50% of patients.[1] However, only a small minority of esophageal lesions will transform into a fistula with the left atrium and even less will lead to cerebral air embolism.[2-9] Since cerebral air or food embolism can be a complication of endoscopy, insertion of nasogastric tubes, and transesophageal echocardiography, these procedures should be contraindicated when there is suspicion of AEF.

Case Report

A 69-year-old man with a history of known cerebrovascular disease was admitted to the neurosciences critical care unit with gram-positive sepsis and symptoms of left-sided weakness and right gaze preference. He was a long-time smoker. His cardiac history was significant for: coronary artery disease, previous myocardial infarction, a left ventricular aneurysm, ischemic cardiomyopathy (ejection fraction 38%), monomorphic ventricular tachycardia with implantable cardioverter-defibrillator (ICD), and atrial fibrillation treated with radiofrequency ablation 50 days prior to admission. On admission, he was on warfarin sodium for anticoagulation of a known left ventricular thrombus and had a supratherapeutic international normalized ratio (INR) of 4.1. The warfarin sodium was held. Magnetic resonance imaging was

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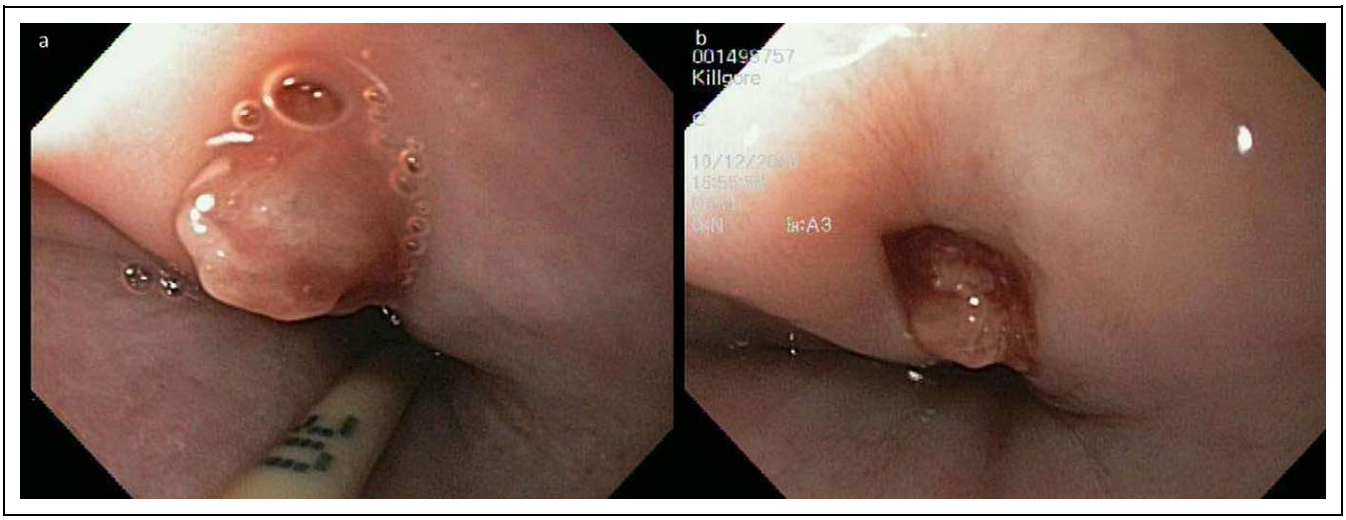

Figure 1. Upper endoscopy demonstrating nodular lesion in upper 1/3 of esophagus (a). Repeat upper endoscopy revealing nodular lesion with surrounding blood (b). Images were obtained from report by permission of gastrointestinal (GI) division at the University of Utah.

not obtainable due to his ICD. Consequently, head computed tomography (CT) with perfusion imaging demonstrated decreased cerebral blood flow in the right anterior cerebral artery and middle cerebral artery watershed regions suggesting ischemia and explaining the neurological findings. Speciation of blood cultures revealed *Streptococcus salivarius*, which raised concern for a contaminant or a gastrointestinal source. The patient was treated with intravenous vancomycin

On the second hospital day, he suffered from a partial complex seizure, became disoriented, and his breathing became labored requiring intubation for airway protection. Continuous electroencephalography monitoring revealed no evidence of epileptiform activity, but he was treated with 1000 mg of levetiracetam twice daily without evidence of further seizure activity. He remained intubated on minimal ventilator settings for airway protection the remainder of his hospital stay as his mental status did not improve.

Because of his stroke and left ventricular thrombus, the patient was restarted on anticoagulation with intravenous unfractionated heparin due to subtherapeutic INR and warfarin sodium was resumed. Subsequently, on hospital day 7, the patient had melena and his hematocrit dramatically dropped from 29.3 to 19.9 with a partial thromboplastin time of 56 and an INR of 1.5. The patient's stool was melenic and positive for blood products. The patient was placed on an esomeprazole drip, the anticoagulation was stopped, he was given 5 mg oral vitamin K, and he underwent upper endoscopy which demonstrated nodular tissue protruding into the esophagus with no bleeding (Figure 1a). The tissue appeared to be "poking" through a small hole in the esophagus which raised concern for atrial-esophageal fistula. The patient remained stable for the next several days off all anticoagulation.

The clinical challenge at this time was to determine when to restart anticoagulation in this patient who was at high risk for additional thromboembolic strokes secondary to the left ventricular thrombus, but whom had developed a significant gastrointestinal (GI) bleed and high suspicion of atrial-esophageal fistula. On hospital day 10, the patient underwent cardiac CT. This study demonstrated an amorphous area of decreased attenuation from the posterior wall of the left atrium to immediately anterior to the thoracic esophagus, which was worrisome for an anomalous communication.

Also on hospital day 10, the patient's neurological exam demonstrated a new right upper extremity plegia. This was concerning for new stroke as he was off anticoagulation due to GI bleeding. Repeat noncontrast head CT demonstrated several new left-sided foci of low density consistent with interim ischemia. Anticoagulation was restarted in an effort to minimize neurological devastation. However, over the next day, he continued to drop his hematocrit and the melenic stools resumed. A second upper endoscopy was performed on hospital day 12 revealing blood oozing from the nodular lesion in the upper third of the esophagus along with rhythmic movement in concert with left atrial filling and ventricular contraction (Figure 1b). This finding was discussed again with cardiothoracic surgery and cardiology for possible intervention. Due to his high risk of intraoperative mortality, they felt he was not a surgical candidate and he was instead scheduled for esophageal stent placement.

Unfortunately, secondary to air introduction from the endoscopy, his neurological status quickly deteriorated. He sustained loss of brainstem reflexes, and emergent noncontrast head CT revealed extensive multifocal intravascular pneumocephalus within the cortical vessels, dural venous sinuses, subarachnoid space, and within the cerebral parenchyma (Figure 2).

The patient died early the next morning after a family discussion where the decision was made for extubation and comfort measures. Autopsy revealed an AEF (Figure 3a-c) as well as extensive air emboli in cerebral vessels (Figure 3d).

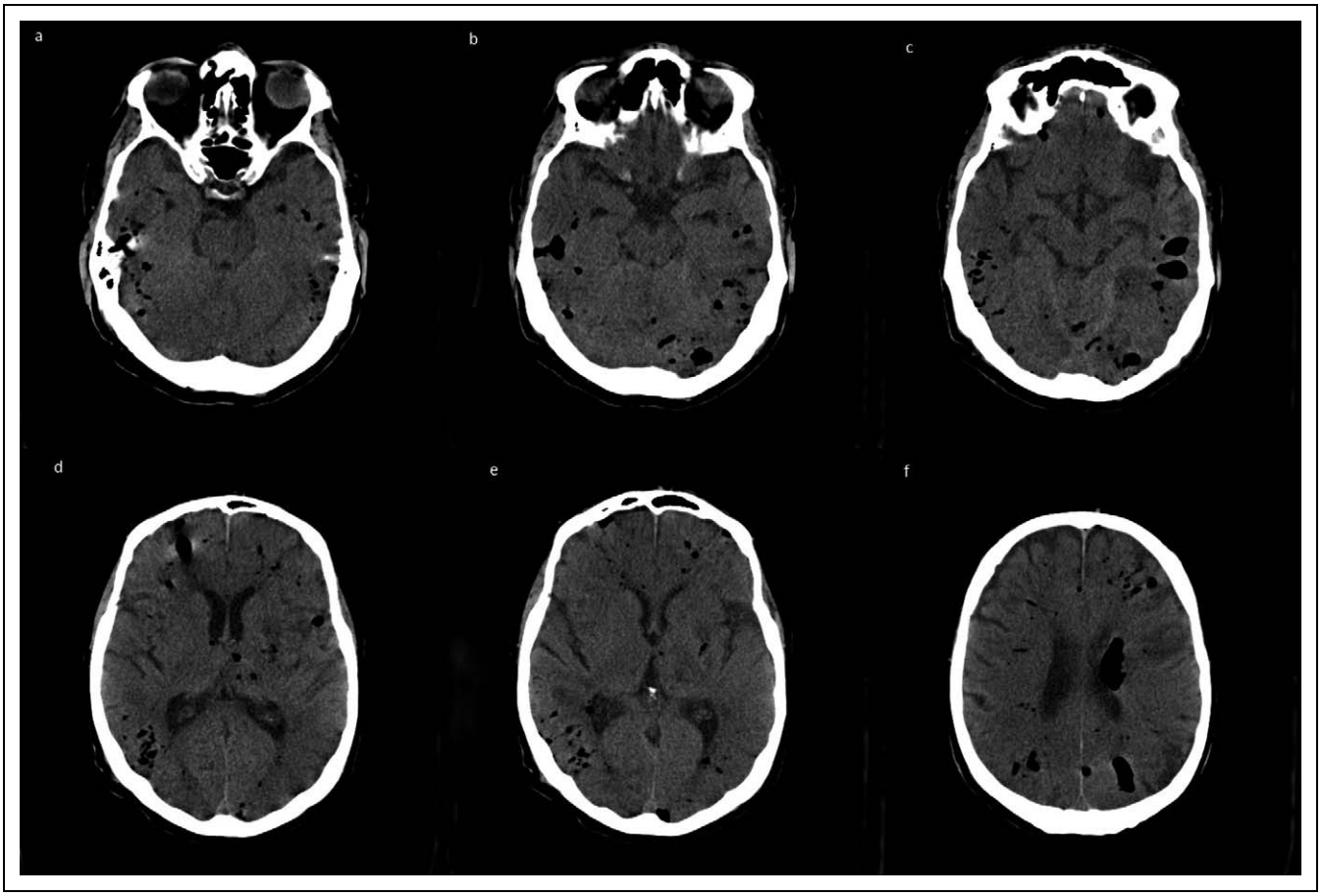

Figure 2. Noncontrast head computed tomography (CT) on the patient on hospital day 12 demonstrating extensive, multifocal intravascular pneumocephalus within the cortical vessels, dural venous sinuses, subarachnoid space, and within the cerebral parenchyma.

Discussion

Our case is the first to demonstrate a delay in presentation of AEF as long as 50 days after the procedure. The case also demonstrated cerebral air emboli as a severe consequence after upper endoscopy in a patient with a suspected AEF. The usual time course has been reported from within a few days to 41 days following the procedure. With the prolonged timeframe and absence of the typical catastrophic presentation in our patient, the diagnosis of AEF was considered to be less likely as the cause of the GI bleed, but ultimately manifested through air embolism and consequent neurological devastation. Suspicion of AEF prior to upper endoscopy is important to avoid direct introduction of air through the fistula and other methods of investigation can be utilized instead.

In the literature, 1 case report describes a patient with esophageal atrial fistula (EAF) associated with esophageal carcinoma who presented with cerebral air emboli several months after radio-ablation of the esophagus with subsequent injury to the left atrium.[2] Another case report describes air embolism after 41 days but was a patient who had a known esophageal injury that was diagnosed 10 days after the procedure.[3]

Ablation for atrial fibrillation has been performed since the 1980s and has mostly entailed atrioventricular (AV) nodal ablation. It is considered to be a safe and effective method for treatment of medically refractory atrial fibrillation. In the last 10 years, pulmonary vein isolation (PVI) has become the most frequently used method. It is performed by catheter radio-ablation and has been associated with low risk of complications.[4-6] Known structural complications include pulmonary vein stenosis and cardiac tamponade, as well as vagal nerve injury with each occurring in up to 1% of cases.[1]

Atrial-esophageal fistula is a more recently discovered problem and PVI has a reported association with AEF in 0.5% to 1% of cases,[5] with other studies demonstrating a lower incidence of 0.01 to 0.2%.[7] Nearly 50 case reports have been described since 2001. Esophageal injury during the procedure is a known complication of ablation of atrial fibrillation and is due to the close proximity between the left atrium and the esophagus with autopsy studies demonstrating a distance often as small as 5 mm.[10] There are esophageal wall changes such as simple erythema to tissue necrosis and ulcers that have been described by endoscopy in up to 47% of

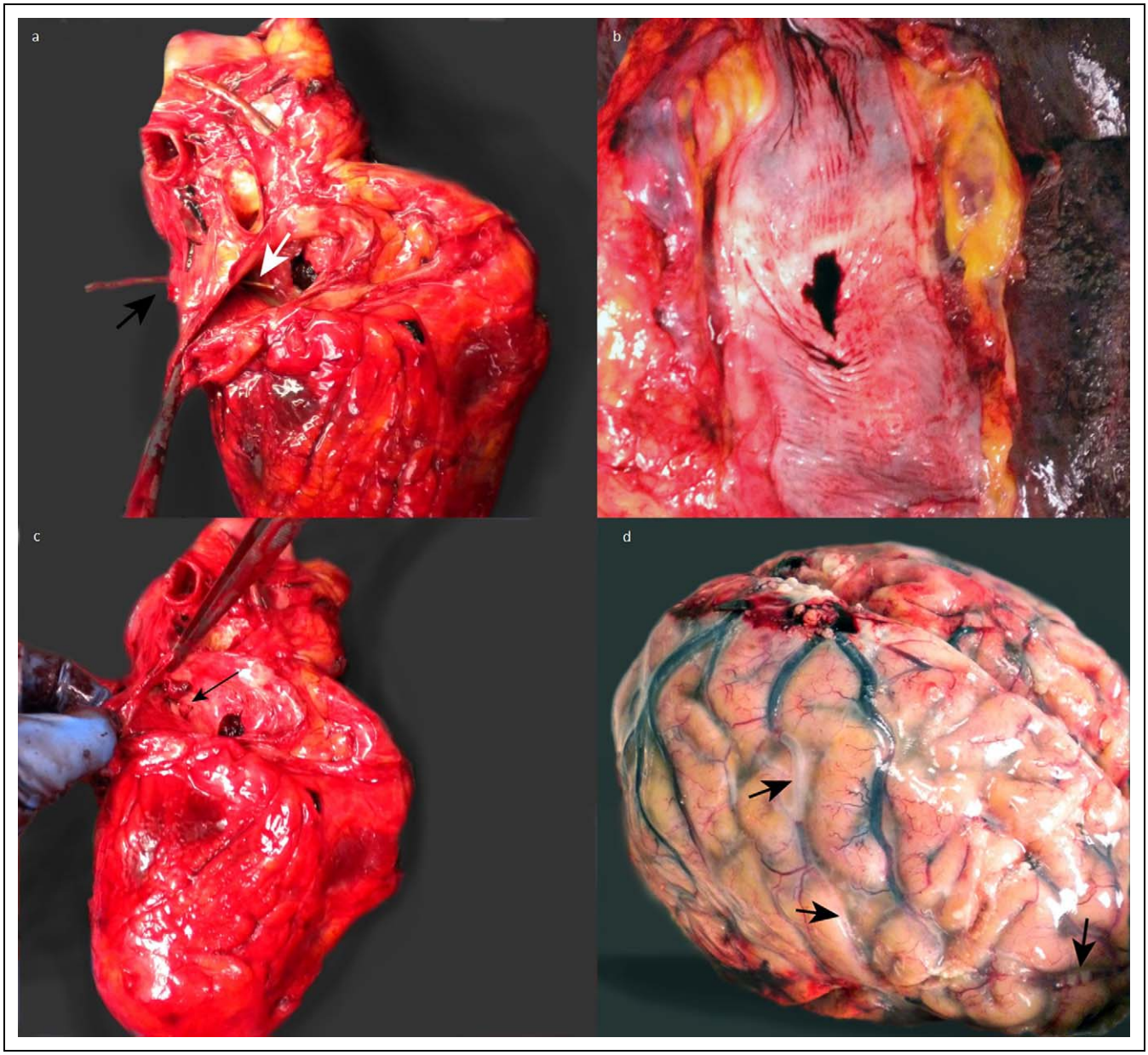

Figure 3. Esophageal side; opening of the esophagoatrial fistula into the left atrium (a). Esophagoatrial fistula with black arrow demonstrating entrance of probe into esophagal side of fistula, and the white arrow demonstrating the probe passing through the atrial side of the fistula (b). Atrial side; black arrow demonstrating the atrial opening of the esophagoatrial fistula into the esophagus (c). Pneumocephalus at autopsy. Black arrows demonstrating air emboli in the superficial vessels (d).

patients following PVI. The formation of AEF is still uncommon even with esophageal injury.[8] The mortality associated with AEF has been reported to be 67% to 100%.[8,9]

More recently, intraoperative measures have been undertaken to reduce the risk of AEF. Intraluminal esophageal temperatures are often monitored during ablation. High temperatures are reached during the radiofrequency ablation procedure and it has been reported that intraluminal temperature less than 41°C is associated with a decreased incidence of esophageal ulcer formation.[11] Further safeguards include a reduction in the power of radio-ablation as well as the duration of ablation, which have been shown to decrease the depth of esophageal lesions as well as the area of esophageal involvement.[12] Due to the high incidence of esophageal injury and potential severity of complications, 1 study suggested follow-up with EGD and endosonography for reevaluation of esophageal injury postablation.[13] However, if AEF is already suspected, it has also been suggested that esophageal procedures be completely avoided due to air embolization as a direct cause from endoscopy.[8] Further safeguards include a reduction

in the power of radio-ablation as well as the duration of ablation which have been shown to decrease the depth of esophageal lesions as well as the area of esophageal involvement.[12]

Our patient had a complicated cardiac history which made surgical treatment of the fistula difficult. The need for intervention was critical in this case and earlier diagnosis of the fistula may have proved life-saving. Our case highlights the need for effective communication between medical specialties concerning tough decisions that determine the care of the patient. Furthermore, with suspicion of AEF, endoscopy should be contraindicated and imaging methods such as cardiac CT are preferred.

Finally, timely consideration of atrial esophageal fistula in patients presenting with new neurological deficits up to 7 weeks postablation may allow for earlier diagnosis and treatment of this rare but fatal entity. With the increased frequency of radiofrequency ablation used to treat atrial fibrillation, emphasis on the welfare of the patient through both intraoperative safety measures as well as enhanced awareness of this complication are essential.

Declaration of Conflicting Interests

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